ORIGINAL RESEARCH

Glucose Infusion into Exercising Dogs After Confinement: Rectal and Active Muscle Temperatures

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Background: Intravenous glucose infusion into ambulatory dogs results in attenuation of exercise-induced increase of both rectal and thigh muscle temperatures. ***Hypothesis:*** That glucose (Glu) infusion attenuates excessive increase in body temperature from restricted activity during confinement deconditioning. ***Methods:*** Rectal (T_{re}) and quadriceps femoris muscle (T_{mu}) temperatures, metabolic rate, and blood samples were taken before and after 90 min of moderate treadmill exercise ($\bar{X}$ = 3.1 ± SE 0.2 $W \cdot kg^{-1}$) at T_{db} = 21 ± 1°C and 45–60% rh from 7 male mongrel dogs (19.6 ± SD 3.0 kg) with i.v. infusion of 40% Glu in 0.9% NaCl (0.07 $ml \cdot kg^{-1} \cdot min^{-1}$) or 0.9% NaCl (0.07 $mg \cdot g^{-1} \cdot min^{-1}$) both before and after 8 weeks of cage confinement. ***Results:*** Mean (±SE) ΔT_{re} (90-0 min) were: NaCl · after = 1.8 ± 0.4°C vs. 1.4 ± 0.3°C (NS) before confinement; Glu · after = 1.3 ± 0.2°C vs. 0.9 ± 0.3°C ($p < 0.02$) before confinement. Comparable ΔT_{mu} (90-0 min) data were: NaCl · after = 2.5 ± 0.4°C vs. 1.9 ± 0.4°C (NS) before; Glu · after = 1.6 ± 0.2°C vs. 1.4 ± 0.4°C (NS) before. Glucose infusion significantly attenuated the rise of T_{mu} (1.9° vs. 1.4°C) only before confinement, but attenuated the rise of T_{re} both before (1.4° vs. 0.9°C) and after (1.8° vs. 1.3°C) confinement. Body temperature attenuation was not related to change in plasma volume, osmolality, [Glu], [lactate], [cortisol], or heat production with constant $\dot{V}O_2$. ***Conclusion:*** Intravenous glucose infusion attenuates the rise in exercise core temperature in deconditioned dogs by a yet undefined mechanism.

EXERCISE BODY temperature and thermoregulatory responses are modified in men and women after thermoneutral water immersion (5) and prolonged bed rest deconditioning (1,4), and in male dogs following prolonged confinement (9,10,11). The rate of increase (1,9,10) and equilibrium level (4,10) of body core temperature in deconditioned humans and confined male dogs are elevated excessively (greater than normal) during exercise when compared with ambulatory-control response, and probably also in deconditioned astronauts.

Turlejska and Nazar (12) found excessive increase (by 0.4°C) in rectal temperature of exercising dogs when glucose metabolism and utilization were attenuated via insulin-induced hypoglycemia and infusion of 2-deoxy-D-glucose. Subsequently, Kruk et al. (8) found that intravenous infusion of glucose into normal-ambulatory dogs resulted in attenuation (by 0.9°C) of exercise-induced increase of both rectal and thigh (active) muscle temperatures, which were accompanied by 16% reduction in oxygen uptake and elevated respiratory exchange ratio, indicating increased carbohydrate metabolism. Therefore, decreased and increased carbohydrate availability appear to affect exercise thermoregulation in dogs.

Thus, the purpose of this study was to test whether intravenous glucose infusion attenuates the excessive rise in exercise-induced rectal and active muscle temperatures after prolonged confinement-deconditioning in dogs.

METHODS

This study was approved by the research review committee of the Polish Academy of Sciences Department of Applied Physiology. The dogs, housed in an indoor kennel and exercised regularly, were fed a nutritionally adequate diet composed of cereals, beef broth, vegetables, and meat scraps during control and confinement periods. The dogs' health was examined weekly by a veterinarian and their humane treatment was checked frequently by personnel from the Polish Animal Care Society.

Procedure: Rectal (T_{re}) and quadriceps femoris muscle (T_{mu}) temperatures, metabolic rate, heart rate, and blood samples were obtained during two randomized experimental treatments (NaCl infusion and glucose infusion) from 7 male mongrel dogs weighing 15.4–23.6 kg ($\bar{X}$ 19.6 ± SD 3.0 kg) before and after 8 weeks of cage confinement. They performed 90 min of moderate treadmill exercise (12° slope, 1.4–1.6 $m \cdot s^{-1}$, 3.1; ± SE 0.2 $W \cdot kg^{-1}$) with ambient temperature of 21 ± 1°C and 45–60% relative humidity. The cages were 40 cm wide, 80 cm high,

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and 110 cm long, which limited movement to standing, lying down, and slight forward and backward movement; the dogs could not turn around. The exercise load after confinement ($W \cdot kg^{-1}$) was recalculated from the post-confinement body weight to equal the pre-confinement load. Before and after confinement the two experimental treatments during exercise were continuous cephalic intravenous infusion of 40% glucose in 0.9% NaCl (30 mg of glucose in 0.07 mL infused at 0.07 $mL \cdot kg^{-1} \cdot min^{-1}$) or 0.9% NaCl (0.07 $mg \cdot kg^{-1} \cdot min^{-1}$) solutions, with at least 5 d between treatments when the dogs remained confined after confinement. The volume of infused fluid was 97–212 mL. Each animal was accustomed to running on the treadmill with its head in a ventilated plastic helmet. Before each experiment the dogs were deprived of food for 20–24 h, but they had free access to drinking water.

Physiological measurements: Rectal and quadriceps femoris muscle temperatures were measured with thermistors and recorded from an electrothermometer (Ellab, Copenhagen, Denmark). The rectal probe was inserted 8 cm and readings were taken before (0 min) and at 15-min intervals during exercise. Muscle temperature was measured before exercise and at 30-min intervals during exercise with a needle thermistor (inserted 2.5 cm) which was removed after each measurement.

Oxygen uptake and CO_2 production were measured at 15-min intervals from the exhaled air stream (200 $L \cdot min^{-1}$) using a Spirolyt II (UEB, Jukalor, Germany) gas analyzer. Heart rate was determined from ECG R-R intervals each 15-min.

Blood measurements: Venous blood samples (25 mL) were taken via an indwelling catheter in a cephalic vein twice before and immediately after (90 min) exercise.

Microhematocrit (Hct) tubes were spun for 3 min at 13,000 rpm and read on a model 36-1 tube reader (Poland). Plasma osmotic concentration [Osm] was determined by freezing point depression with a Fiske osmometer (model 9-66, Bethel, CT). Blood glucose [Glu] and lactate [La] concentrations were measured with enzymatic methods using commercial kits (Boehringer Diagnostica, Mannheim, Germany), and plasma cortisol was determined by radioimmunoassay with antibodies from the Polish Academy of Sciences Institute of Animal Physiology—Jablonna.

Statistical analyses: The data were analyzed with two-way analysis of variance, Student's *t*-test for paired samples, and the Wilcoxon matched pairs test for the 90 min temperature data. The null hypothesis was rejected when $p < 0.05$, and nonsignificant (NS) differences are referred to as trends or tendencies.

RESULTS

Physiological measurements: Mean resting (0 min) oxygen uptake before confinement for the NaCl (19.6 ± SE 1.3 $mL \cdot min^{-1} \cdot kg^{-1}$) and glucose (19.6 ± 1.8 $mL \cdot min^{-1} \cdot kg^{-1}$) treatments was reduced significantly after confinement to 14.0 ± 1.7 ($t = 5.29$, $p < 0.01$) and 14.3 ± 0.4 $ml \cdot min^{-1} \cdot kg^{-1}$ ($t = 3.02$, $p < 0.05$), respectively (**Fig. 1,** upper panel). There were no differences in the significantly increased ($p < 0.0001$) oxygen uptake of all four treatments during exercise to between 39.0 ± 1.4 and 42.6 ± 1.3 $ml \cdot min^{-1} \cdot kg^{-1}$.

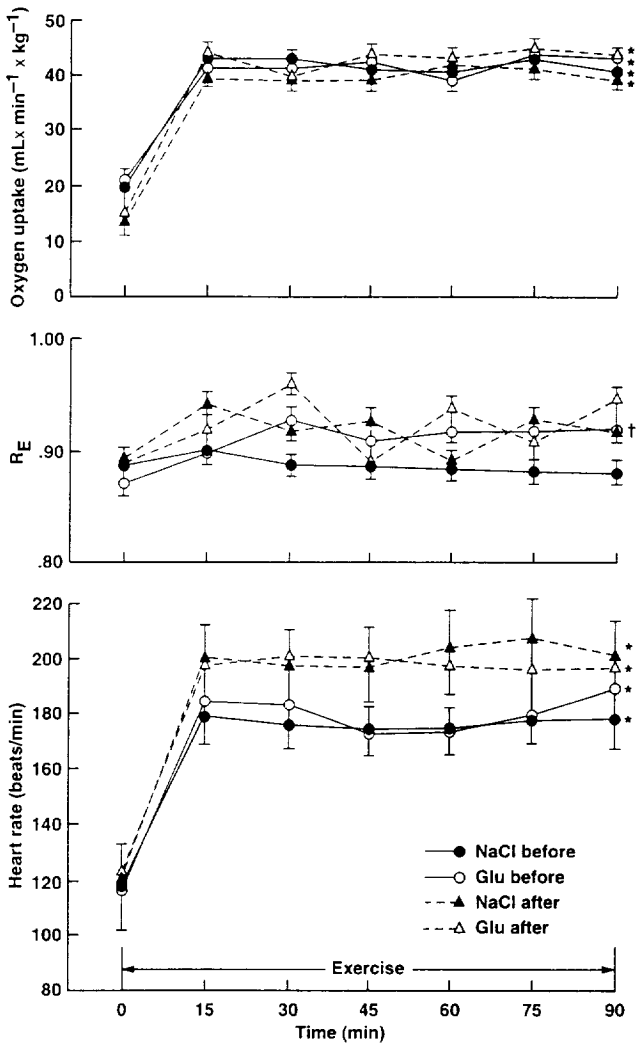

Fig. 1. Mean (±SE) oxygen uptake, respiratory exchange ratio (RE), and heart rate at rest (0 min) and exercise with NaCl and glucose (Glu) infusion before and after 8-wk confinement. * $p < 0.05$ from 0 min, † $p < 0.05$ from NaCl · before.

Mean respiratory exchange ratios (R_E) for all treatments varied from 0.87 ± 0.04 to 0.89 ± 0.04 (NS) at rest, and were not different during exercise except Glu · before (0.91 ± 0.01) was greater ($p < 0.025$) than NaCl · before (0.86 ± 0.01) (Fig. 1, middle panel). The ratio increased at 90 min above NaCl · before (0.84 ± 0.04) to 0.92 ± 0.04 ($p < 0.05$) with Glu · before and to 0.95 ± 0.03 ($p < 0.05$) with Glu · after, which would be expected with glucose infusion.

Mean resting heart rate varied from 116 ± 15 to 122 ± 10 $beats \cdot min^{-1}$ (NS) with all treatments (Fig. 1, lower panel). Exercise heart rate for all treatments increased ($p < 0.0001$) to approximately 180 $beats \cdot min^{-1}$ (NaCl · before and Glu · before) with trends (NS) to about 200 $beats \cdot min^{-1}$ with NaCl · after and Glu · after confinement, which would also be expected.

Mean absolute and change (90 min-0 min) in rectal and muscle temperatures pre- and post-exercise with both infusions are presented in **Table I.** Mean change in rectal temperature at 90 min with all treatments was elevated significantly ($p < 0.0001$) during exercise (**Fig. 2,** upper

TABLE I. MEAN (±SE) ABSOLUTE AND CHANGE IN RECTAL AND MUSCLE TEMPERATURES (°C) PRE (0 MIN) AND POST (90 MIN) EXERCISE WITH NaCl AND GLUCOSE INFUSIONS BEFORE AND AFTER 8 WEEKS OF CAGE CONFINEMENT IN 7 DOGS.

	NaCl			Glucose			
	0	90	Δ	0	90	Δ	Δ NaCl vs. Δ Glucose
Rectal Before Confinement							
$\bar{X}$	39.2	40.6	1.4	39.2	40.1	0.9	<0.028
±SE	0.1	0.3	0.3	0.1	0.3	0.3	
Rectal After Confinement							
$\bar{X}$	39.2	41.0	1.8†	39.3	40.6	1.3†	<0.028
±SE	0.2	0.4	0.4	0.1	0.3	0.2	
Muscle Before Confinement							
$\bar{X}$	39.3	41.2	1.9	39.4	40.8	1.4	<0.028
±SE	0.1	0.4	0.4	0.1	0.4	0.4	
Muscle After Confinement							
$\bar{X}$	39.4	41.9	2.5†	39.6	41.2	1.6	<0.028
±SE	0.2	0.3	0.4	0.1	0.2	0.2	

† $p < 0.028$ to <0.043 vs. comparable Δ before confinement.

panel); after confinement T_{re} was higher than before confinement: NaCl · after = 1.8 ± 0.4°C vs. 1.4 ± 0.3°C before ($p = 0.043$),and Glu · after = 1.3 ± 0.2°C vs. 0.9 ± 0.3°C before ($p = 0.018$). At 90 min the ΔT_{re} for Glu · before (0.9°C) was significantly lower ($p = 0.028$) than that for NaCl · before (1.4°C); and Glu · after (1.3°C) was significantly lower ($p = 0.028$) than that for NaCl · after (1.8°C). Thus, glucose infusion significantly attenuated the rise of T_{re} both before and after 8 weeks of confinement.

Increase in all muscle temperatures ($p < 0.0001$) during exercise followed rectal temperatures qualitatively (Fig. 2, lower panel); i.e,. NaCl · after T_{mu} had the greatest increase (Δ = 2.3°C), followed by NaCl · before (Δ = 1.9°C), Glu · after (Δ = 1.6°C), and Glu · before (Δ = 1.4°C). Changes in all T_{mu} at 90 min were about 0.5°C higher than the comparable ΔT_{re} (**Fig. 3**). Because Glu · after ΔT_{mu} (1.6°C) was not significantly different from Glu · before ΔT_{mu} (1.4°C), the ΔT_{mu} for the four treatments was not as differentiated as that for ΔT_{re}, but glucose infusion significantly attenuated the increased muscle temperature before but not after confinement.

The increase in rectal and muscle temperatures for the four treatments were related linearly and significantly ($r = 0.91$, $p < 0.001$), with ΔT_{mu} consistently higher than ΔT_{re} (Fig. 3). The difference from equality was 0.14°C at rest (Y-intercept) which increased progressively with increasing temperatures.

Blood measurements: Plasma [cortisol] was not significantly different after exercise (90 min) with all treatments (**Table II**). It was unchanged after exercise with NaCl · before; but tended to increase by 18–28% (NS) after exercise with the three other treatments suggesting increased strain, especially after confinement and with glucose infusion.

Hematocrit and plasma osmotic concentration were not different after confinement (Table II). Mean raw Hct before exercise for the four treatments ranged from 49.3–51.2% (NS), while post-exercise values tended to increase by 1–4 units (NS) suggesting exercise-induced hemoconcentration (assuming no change in RBC mass). In like manner, [Osm] before exercise were not different and tended to increase by 0–3% (NS) with all treatments after exercise.

Plasma [Glu] before confinement decreased from 3.35 pre-exercise to 2.81 $mM \cdot L^{-1}$ post-exercise (Δ = −16%, $p < 0.015$), while post-exercise [Glu] from the other three treatments were not different (Table II). Before and after confinement, post-exercise [Glu] tended to decrease (NS) with NaCl and to increase by 3–11% (NS) with glucose infusion, as expected. On the other hand, post-exercise [La] increased significantly by 115–304% ($p < 0.05$) with all four treatments with no apparent effect of infusate (Table II).

DISCUSSION

Core temperature response in the present study confirmed that from our prior 8-week confinement data in dogs without infusion (9–11) where excessive increase in exercise T_{re} after confinement was above the ambulatory control level by 0.7°C (10) and by 1.3°C (9). The excessive increase in T_{re} at 90 min of treadmill exercise (3.1 $W \cdot kg^{-1}$) after confinement in the present study (with NaCl infusion) was not significantly different from the mean T_{re} from our prior confinement study with similar parameters (10 d, 15–25 kg, 1.4–1.6 $m \cdot s^{-1}$ = 3.3 $W \cdot kg^{-1}$ also at 12° inclination) without NaCl infusion (9). Present study 90 min mean (±SE) T_{re} and ΔT_{re} before confinement were 40.5 ± 0.3°C and 1.3 ± 0.3°C, respectively; comparable data from reference 8 were 40.4 ± 0.6°C (NS) and 1.3 ± 0.2°C (NS), respectively: after confinement data were 41.0 ± 0.4°C and 1.8 ± 0.3°C (present study) compared with 41.5 ± 0.3°C (NS) and 2.6 ± 0.3°C ($p < 0.01$) from our prior study (9). Thus, while there was no effect

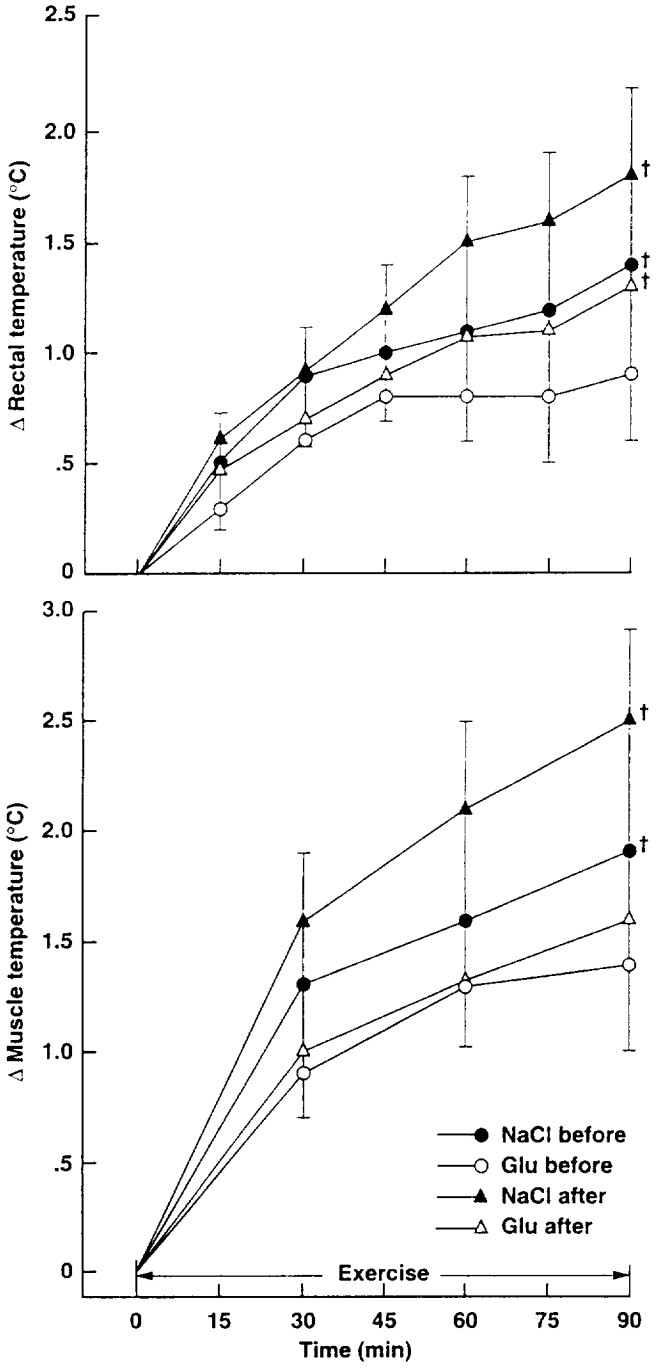

Fig. 2. Mean (±SE) change in rectal and quadriceps femoris muscle temperatures during exercise with NaCl and glucose (Glu) infusion before and after 8-wk confinement. † $p < 0.05$ from Glu · before.

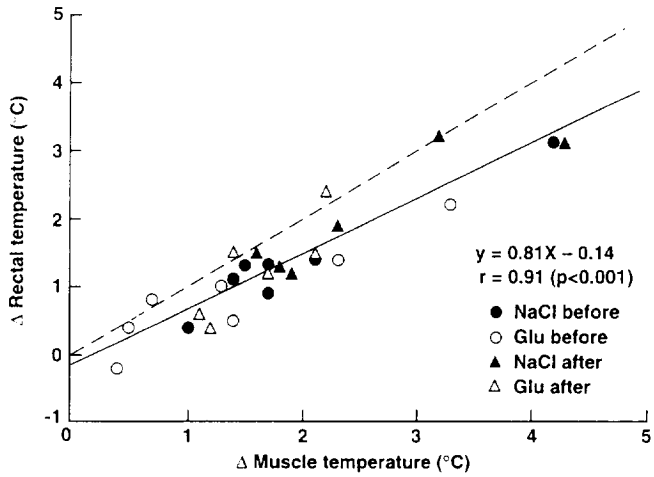

Fig. 3. Regression of mean increase in rectal temperature on increase in muscle temperature at 90 min of exercise with NaCl and glucose (Glu) infusion before and after 8-wk confinement. (- - - -) = Line of identity.

of NaCl infusion on exercise ΔT_{re} before confinement, infusion appeared to attenuate ΔT_{re} by about 0.8°C after confinement. This attenuation would appear to be a net result of the hyperthermic effect of the improbable increase in plasma sodium and osmotic concentrations (3,7), and the hypothermic effect of increased plasma volume (2) from the infusate (volume = 9 mL · kg^{-1}). The latter seems to dominate.

The more important findings from the present study were the significantly attenuated increase in exercise T_{re} with glucose infusion both before and after confinement, and in T_{mu} *before* but not after confinement. Turlejska and Nazar (12) found excessive increase in T_{re} in exercising dogs pretreated with insulin or 2-deoxy-D-glucose (a competitive inhibitor of glucose utilization), which decreased availability of glucose in the central nervous system resulting in release of hormones (e.g., catecholamines) which stimulate metabolic rate. On the other hand, Kruk et al. (8) measured rectal and muscle temperatures and thermoregulatory responses in ambulatory exercising (2.2–3.3 W · kg^{-1}) dogs and found that glucose infusion attenuated the rise in both T_{re} (by 0.9°C) and T_{mu} (by 1.0°C). These attenuated temperatures were accounted for by a combination of overall decreased heat production (oxygen uptake) and increased heat dissipation; i.e., increased thermoregulatory efficiency which may have been due, in part, to increased plasma volume from infusion.

Thermoregulatory efficiency, a function of rate of total body heat production and dissipation reflected in level of body temperature, is under control of hypothalamic thermoregulatory centers. In the present study there was a trend toward higher mean oxygen uptake with glucose infusion, especially after confinement, so depressed metabolic rate and heat production cannot account for the attenuated core temperatures before and after confinement. Plasma [Glu] during exercise after confinement tended to be more stable and even to increase slightly with glucose infusion, indicating adequate substrate availability to support the increased exercise metabolism. The respiratory exchange ratio was higher (0.92–0.94) during glucose infusion as expected, when compared with NaCl infusion, indicating greater carbohydrate metabolism. Plasma [La] increased significantly with all treatments during exercise, suggesting no major impairment in exercise substrate metabolism. Thus, reduced carbohydrate availability and metabolism, or lower gross metabolic heat production, cannot account for the attenuated temperatures (6).

It appears, from the intermingling of the T_{re} and rela-

TABLE II. MEAN (±SE) HEMATOCRIT, PLASMA OSMOLALITY AND BLOOD GLUCOSE, LACTATE, AND CORTISOL CONCENTRATIONS PRE (0 MIN) AND POST (90 MIN) EXERCISE WITH NaCl AND GLUCOSE INFUSIONS BEFORE AND AFTER 8 WEEKS OF CAGE CONFINEMENT IN 7 DOGS.

	NaCl				Glucose			
	0	90	%Δ	P	0	90	%Δ	P
Cort Before Confinement ($\mu g \cdot dl^{-1}$)								
$\bar{X}$	4.3	4.3	0	0.992	4.0	4.7	18	0.726
±SE	0.6	1.0			0.6	1.6		
Cort After Confinement								
$\bar{X}$	50.8	65.2	28	0.363	36.1	45.6	26	0.154
±SE	14.3	13.4			11.2	12.1		
Hct Before Confinement (%)								
$\bar{X}$	49.7	51.6	4	0.154	48.5	50.5	4	0.109
±SE	2.1	1.5			1.8	1.2		
Hct After Confinement								
$\bar{X}$	51.2	52.5	3	0.189	49.3	49.7	1	0.658
±SE	1.2	0.6			1.4	1.4		
Osm Before Confinement ($mOsm \cdot kg^{-1}$)								
$\bar{X}$	285	288	1	0.144	287	288	0	0.195
±SE	6	9			10	9		
Osm After Confinement								
$\bar{X}$	295	295	0	0.100	286	295	3	0.537
±SE	2	6			6	8		
Glu Before Confinement ($mM \cdot L^{-1}$)								
$\bar{X}$	3.35	2.81	−16	0.015*	3.37	3.48	3	0.671
±SE	0.17	0.18			0.31	0.28		
Glu After Confinement								
$\bar{X}$	3.64	3.30	−9	0.116	3.66	4.07	11	0.389
±SE	0.26	0.30			0.32	0.44		
La Before Confinement ($mM \cdot L^{-1}$)								
$\bar{X}$	1.71	3.68	115	0.002*	1.77	4.51	155	0.004*
±SE	0.15	0.40			0.10	0.62		
La After Confinement								
$\bar{X}$	1.34	5.42	304	0.001*	1.33	3.62	172	0.001*
±SE	0.18	0.50			0.14	0.18		

* $p < 0.05$ for 90-min vs. 0-min.

tively higher T_{mu} data points at 90 min of exercise (Fig. 3), that there was no selective effect of glucose infusion that would cause a shift in the set-point of the central controller to attenuate temperature increases while maintaining the 0.4°C temperature difference between before and after confinement levels (except for the 0.2°C T_{mu} difference with glucose infusion). Increased plasma ion-osmotic concentrations, independent of change in plasma volume, appear to influence (perhaps control) exercise thermoregulation by direct effect on brain thermoregulatory centers (2,3). Elevated plasma osmolality, independent of plasma [Na^+], is associated with significantly higher exercise T_{re} in ambulatory dogs (7). In the present study the Hct, with consideration for splenic entrapment of RBC (and plasma volume by inference), and plasma osmolality with all treatments were not significantly different during exercise; so these variables were probably not involved in the temperature attenuation.

The excessive increase in core temperature in humans after prolonged bed-rest-deconditioning appears to be caused by attenuation of conductive heat dissipation via reduced peripheral vasodilation that was not necessarily associated with hypovolemia or lower evaporative heat loss via reduction in sweating (1,4). Reduced heat loss

in dogs would involve mainly attenuated evaporative water loss via respiratory rate and volume which remain to be measured. If it can be shown that increased plasma glucose results in attenuation of the deconditioning-induced excessive exercise-induced hyperthermia in humans, then perhaps an appropriate intravenous solution could be formulated to attenuate any significant hyperthermia of astronauts working in microgravity.

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